Published in final edited form as:
J Immigr Minor Health. 2013 October ; 15(5): 910–917. doi:10.1007/s10903-012-9690-7.

Self-Reported Prior Lung Diseases as Risk Factors for Non-small Cell Lung Cancer in Mexican Americans

Michelle K. McHugh,
Department of Epidemiology, The University of Texas MD Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030, USA

Matthew B. Schabath,
Department of Cancer Epidemiology, H. Lee Moffitt Cancer Center and Research Institute, Tampa, FL 33612, USA

Chung-han Ho,
Department of Epidemiology, The University of Texas MD Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030, USA

Mei Liu,
Department of Epidemiology, The University of Texas MD Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030, USA

Anthony M. D'Amelio Jr.,
Department of Epidemiology, The University of Texas MD Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030, USA

Anthony J. Greisinger,
Kelsey Research Foundation, Houston, TX 77005, USA

George L. Delclos,
Division of Epidemiology, Human Genetics and Environmental Sciences, The University of Texas Health Science Center at Houston School of Public Health, Houston, TX 77030, USA. Universitat Pompeu Fabra, Barcelona, Spain. CIBER Epidemiología y Salud Pública (CIBERESP), Center for Research in Occupational Health, Barcelona, Spain

Margaret R. Spitz, and
Dan L. Duncan Cancer Center, Baylor College of Medicine, Houston, TX 77030, USA

Carol J. Etzel
Department of Epidemiology, The University of Texas MD Anderson Cancer Center, 1515 Holcombe Boulevard, Houston, TX 77030, USA

Carol J. Etzel: cetzel@mdanderson.org

Abstract

This study was conducted to assess the association between prior history of respiratory disease and lung cancer among Mexican Americans using data from a multi-racial/ethnic lung cancer case–control study. Cases (n = 204) were patients with previously untreated lung cancer. Healthy control participants (n = 325) were recruited from a large physician group practice. Demographics, cigarette use, and history of respiratory disease were collected. Multivariable logistic regression models were used to estimate relative risk. Prior history of COPD (OR = 2.0; 95 % CI 1.2–3.3)

Correspondence to: Carol J. Etzel, cetzel@mdanderson.org.

and pneumonia (OR = 2.2; 95 % CI 1.3–3.6) were associated with an increased risk of lung cancer. These findings illustrate that prior COPD and pneumonia are associated with an increased risk of lung cancer among Mexican Americans. To our knowledge, this is one of largest case–control analyses assessing the role of respiratory disease and lung cancer risk specifically among Mexican-Americans.

Keywords

Lung diseases; Mexican Americans; Case–control studies; Epidemiology; Lung cancer

Introduction

In the United States (US) lung cancer is the leading cause of cancer-related deaths among Hispanic men and the second leading cause of cancer-related deaths among Hispanic women [1]. Compared to non-Hispanic whites in the US, Hispanics have a 33 % lower age-adjusted incidence of cancer and a 38 % lower cancer mortality rate, [2] but have worse survival for non-small cell lung cancer [3, 4]. There is evidence that suggest as Hispanics are assimilated into the US culture their lower cancer rates could potentially dissipate and mimic rates observed among non-Hispanic whites [2, 5]. Unfortunately, few epidemiologic studies for lung cancer have specifically targeted Hispanics or Mexican Americans, the largest Hispanic subgroup.

Cigarette smoking accounts for 30 % of all cancer deaths and an estimated 85 % of lung cancer deaths [6], yet only a fraction (~15 %) of smokers actually develop lung cancer [7]. Nonmalignant lung conditions (i.e., asthma, bronchitis, emphysema, hay fever, tuberculosis, and pneumonia) have been associated with an increased risk of lung cancer [8–16] and the injury and inflammation induced by smoking and chronic respiratory diseases may predispose individuals to carcinogenesis in affected tissues [17–21]. However, the biological role of respiratory disease is not entirely elucidated and data from epidemiologic studies have been inconsistent.

To address the paucity of research regarding the health of Mexican Americans, the US' fastest growing population [22], and to better understand the role of lung cancer factors among Mexican-Americans, particularly smoking and prior history of respiratory disease, we analyzed data from a case–control study of lung cancer comprised of Mexican-American lung cancer cases and healthy controls who resided in the Houston, Texas Metropolitan area. To our knowledge, this is one of largest case–control analyses assessing the role of respiratory disease and lung cancer risk specifically among Mexican-Americans.

Methods

Study Population

The study population, covariates, and data collection procedures have been previously described [23, 24]. Briefly, the data were collected between 1991 and 2010 as a part of a multi-racial/ethnic lung cancer case–control study. Cases (n = 204) were patients who presented at The University of Texas MD Anderson Cancer Center with newly diagnosed, histopathologically confirmed lung cancer and were enrolled in the study before initiation of chemo- or radiation therapy. Patients were excluded from the study if they had received prior chemotherapy or radiotherapy at least 6 months prior to consent. Healthy control participants (n = 325) without a prior history of cancer (except non-melanoma skin cancer) were recruited from the Kelsey-Seybold Clinic, Houston's largest multispecialty physician

group practice. There were no age, gender, ethnicity, or disease stage restrictions on recruitment.

Ethnicity or country of origin was ascertained by the following question and response: “Are you of Spanish, Hispanic or Latino ethnicity or origin?” and “Yes, Mexican or Mexican American.” For our analysis, we focused on all the participants who were self-reported Mexican Americans which represented the majority of all self-reported Hispanics and approximately 6 % of the overall multi-racial/ethnic lung study population. All enrollees signed an informed consent and completed a personal interview; proxy interviews were not conducted. The institutional review boards of The University of Texas MD Anderson Cancer Center and the Kelsey-Seybold Clinic approved the study.

Exposure Assessment

Participants were classified as never smokers (<100 cigarettes in their lifetime), former smokers (quit more than 1 year before lung cancer diagnosis for the cases or 1 year before the interview date for the controls) and current smokers. Smoking variables included duration of smoking, number of cigarettes per day and pack-years smoked (i.e., years smoked times the average number of cigarettes per day divided by 20). For our analysis, smoking status was further categorized as 0<pack-years ≤ 20 (light); 20<pack-years ≤ 40 (moderate) and >40 pack-year (heavy). Among former smokers, we also ascertained age of smoking initiation, age at smoking cessation and years since quitting. Medical co-morbidities included self-reported physician-diagnosed respiratory diseases such as asthma, hay fever, chronic obstructive pulmonary disease (COPD; which included emphysema and chronic bronchitis) and pneumonia. Clinical covariates including stage of disease at diagnosis and histological characteristics were abstracted from the patient medical records of the lung cancer cases.

Statistical Analysis

Pearson’s χ^2 test and Student’s *t* test were used to test for differences in distributions between the cases and controls for categorical and continuous variables, respectively. Unconditional logistic regression analyses were used to calculate odds ratios (ORs) and 95 % confidence intervals (CIs) as estimates of lung cancer risk. A simple yes/no binary variable was used for analysis of the respiratory diseases. To address potential residual confounding, the final logistic regression models were adjusted for age, gender, smoking intensity and self-reported pesticide exposure. Given that we previously found an increased risk of lung cancer for those with self-reported pesticide exposure in the same Mexican American lung study population [25], pesticide exposure was added to the final models. All analyses were performed using SPSS version 19 (IBM software, 2010).

Results

Data from 204 cases with lung cancer and 325 healthy controls were available for this analysis (Table 1). There were no statistically significant differences between the cases and controls in terms of gender, age by gender, family history of first degree relatives with cancer, and self-reported history of asthma or hay fever. Approximately 44 % of the cases were self-reported current smokers and 35 % of the cases were former smokers as compared to 30 and 36 %, respectively, for the controls ($P<0.001$). In general, cases reported heavier smoking histories than controls. Among current smokers, cases smoked 51 pack-years [mean (SD) = 34.3] compared with 25 pack-years [mean (SD) = 22.9] among controls. Among former smokers, cases smoked 41 pack-years [mean (SD) = 32.0] compared with 22 pack-years [mean (SD) = 31.6] among controls. Former smoker cases smoked longer [37.9 years, mean (SD) = 15.5] and quit smoking at a later age [57.5 years, mean (SD) = 12.2]

than controls [24.6 years, mean (SD) = 14.7 and 44.7 years, mean (SD) = 14.3, respectively]. Approximately 23 % of cases reported a history of COPD and 25 % of cases reported a history of pneumonia, compared to 12 and 12 %, respectively, among the controls ($P<0.001$). The most common histology was adenocarcinoma (42.3 %) followed by squamous cell carcinoma (26.5 %) and the majority of the cases presented with stage III (28.1 %) or stage IV (50.6 %) lung cancer.

As expected, we observed statistically significantly increased risks among the smoking-related variables except for years of smoking cessation which was inversely associated with lung cancer risk (Table 2). Overall risk for former smokers was 1.6-fold higher (OR = 1.6, 95 % CI 1.0–2.6) compared to never smokers and 2.4-fold higher for current smokers (OR = 2.4, 95 % CI 1.5–4.0). Among current smokers, moderate smokers (20<packyears ≤ 40) exhibited over a fourfold increased risk (OR = 4.2, 95 % CI 1.7–10.3) compared with a tenfold increased risk (OR = 10.0, 95 % CI 4.0–24.7) for heavy smokers (>40 pack-years). Similar patterns of risks were observed among former smokers but the ORs were attenuated. Furthermore, former smokers who had quit smoking for>10 years had a 60 % decreased risk of lung cancer (OR = 0.4, 95 % CI 0.2–0.7) compared to those who quit smoking for 10 years or less. Moreover, former smokers who quit smoking after age 30 years had nearly a sixfold higher risk of lung cancer (OR = 5.8, 95 % CI 1.2–27.4) compared to those who quit before the age of 30 years.

For the testing of those with respiratory co-morbidities, a previous self-reported physician diagnosis of hay fever (OR = 0.4, 95 % CI 0.2–0.9) was inversely associated with lung cancer. Conversely, self-reported history of those with COPD (OR = 2.0; 95 % CI 1.2–3.3) and those with pneumonia (OR = 2.2, 95 % CI 1.3–3.6) were both associated with a statistically significant increased risk of lung cancer. When we conducted the same main-effect models among never smokers only, the findings for asthma, hay fever, COPD and pneumonia exhibited the effects in the same directions but the odds ratios (ORs) were not statistically significant (data not shown).

Discussion

In this case–control analysis comprised of self-reported Mexican-Americans, we found a prior history of pneumonia and prior history of COPD were both associated with an increased risk of lung cancer; yet we found an inverse relationship for those with a prior history of hay fever.

Previous epidemiology studies have reported increased lung cancer risk associated with prior COPD diagnosis in Caucasian and African American populations [8, 15, 24–28]. However, this is the first analysis reporting this association among Mexican-Americans. COPD, primarily emphysema and chronic bronchitis, is characterized by airflow limitation that is not completely irreversible and this airflow limitation is usually progressive and often associated with an inflammatory response to environmental exposures (e.g., cigarette smoking) [29]. In our analysis, COPD was associated with a significant twofold increased risk for lung cancer (OR = 2.0, 95 % CI 1.2–3.3) and these findings are substantially lower than what we previously reported for African Americans (OR = 6.4, 95 % CI 3.2–12.6) [24] and moderately lower among non-Hispanic whites (OR for emphysema = 2.7; 95 % CI 2.2–3.6) [23] from the same multi-racial/ethnic lung cancer case–control study. A recent meta-analysis of twelve studies found an increased risk for lung cancer associated with a history of pneumonia in both prospective studies (OR = 1.2, 95 % CI 1.0–1.3) and retrospective studies (OR = 2.2, 95 % CI 1.8–2.6) [30]. In contrast, Koshiol and colleagues [31] found that individuals who reported multiple pneumonia diagnosis exhibited a statistically decreased risk of lung cancer (OR = 0.8, 95 % CI 0.6–1.0). Since we did not collect number

of pneumonia diagnoses, we could not perform multiple diagnoses analysis in this study. Presently there are no data to support a biologic role for pneumonia in lung cancer; however, one could speculate that the inflammation involved with pneumonia infection could be attributed to this observed relationship. Pneumonia is commonly caused by viruses or bacteria (i.e., *Steptocococcus pneumonia, Haemphilis influenzae, Legionella pneumophilia* and *Chlamydia pneumoniae*) [32–34] and is associated with pulmonary inflammation. Moreover, inflammation has been proposed as a biological mechanism to explain the increased lung cancer risk for those with lung infections including pneumonia and tuberculosis [17–21]. Mechanistically inflammation has been implicated as a cancer initiator or promoter since inflammation generates reactive oxygen species (ROS) which can result in DNA damage; lead to cell injury and impaired DNA repair capacity; increase cell proliferation; as well as stimulate angiogenesis [21, 35–37]. At present the biological role of pneumonia and lung cancer risk is not well-established.

The association between hay fever and lung cancer risk is unclear as previous studies have found protective effects [38–43], no association [13, 38] and increased risk [13, 38]. In the present study we found a statistically significant inverse effect (OR = 0.4, 95 % CI 0.2–0.9) associated with a prior history of hay fever which is also consistent with our previous findings among non-Hispanic whites [44]. As we previously discussed [44], there are several hypotheses that have been postulated to address the association between hay fever and lung cancer. Some studies propose that hay fever results in an enhanced stimulated immune system and is better able to detect and destroy malignant cells thereby conferring a protective effect [38–43]. The protective effect of hay fever may be explained by the proliferation and use of over-the-counter and prescription allergy medications. These agents include antihistamines, corticosteroids, decongestants, mast cell stabilizer, leukotriene modifiers and bronchodilators, all of which block allergy symptoms through various mechanisms [45]. It is also possible that individuals with allergies will make lifestyle choices to prevent exacerbation of their symptoms such as avoiding certain dietary constituents and/or environments with environmental tobacco smoke, air pollution, or occupational carcinogens [44, 46–49]. Further, as stated in our previous findings [45], we cannot overlook the “immune surveillance hypothesis” a process by which tumors may be suppressed by tumor-associated antigens [50].

Since cigarette smoking is more strongly associated with lung cancer than any other cancer [51], we expected to find sizeable increased risks among current- and former smokers. In our study for both case and control participants, current smokers smoked at levels similar to those reported by Haiman et al. [52] and with our previous study in non-Hispanic Whites [23]. The age a participant quit smoking among former smokers remains an important determinant as the reduction in risk is greater the younger the age of smoking cessation [53]. In this present analysis, former smokers who quit after the age of thirty exhibited a threefold increased risk of lung cancer. This finding was not unexpected since cancer incidence of former smokers, which represent the majority of lung cancers diagnosed, does not return to the rate of never smokers [24, 54]. Due to small sample size, were not able to evaluate the interaction between smoking status and previous lung disease.

Limitations in our study necessitate consideration. We used a hospital-based case recruitment system because we were required by the parent grant to identify lung cancer patients prior to therapy. Therefore, our study is not population-based and we recognize this limitation in control accrual. However, the principle that controls must represent the entire population of non-diseased individuals has been debated. Many epidemiologists argue that the selection of controls should be based on individuals who have the possibility of becoming cases [55, 56]. We believe our controls, who were recruited through a local multi-specialty physician practice in the Houston metropolitan area, would have sought treatment

at MD Anderson Cancer Center if they had developed cancer, thereby meeting the criteria of Rothman and Greenland [55]. We acknowledge that one could argue selection bias may occur and those who declined to participate may have had differing rates of lung disease.

Whilst recall and reporting bias are another potential limitation, studies have validated the use of self-report as a viable means for assessing chemical-related exposures [57–59] as well as chronic diseases. Toren et al. [60] determined that questions regarding self-reported physician-diagnosed asthma, regardless of mode of validation (i.e., against a bronchial challenge test; clinical assessment; and/or no comparison) offer the highest specificity and were the most reliable. Others have found that self-reported data for hypertension to be highly correlated with physician records [61–64]. Moreover, misclassification bias due to language barriers and low levels of literacy can occur in well-validated survey instruments [65]. However, we feel recall of co-morbidities in our study would be minimal since participants are more apt to remember if "a doctor ever told you that you have had any of the following medical conditions?" (e.g., asthma, COPD, pneumonia, and hay fever) than if a proxy was used. If misclassification occurred it would be non-differential and therefore underestimate the association [66]. Even though we adjusted for smoking status, COPD is so strongly associated with tobacco use that we cannot overlook the possibility of residual confounding. Finally, small sample size prohibited testing interaction between smoking and prior respiratory disease.

Despite these limitations these data suggest that prior respiratory diseases such as COPD and pneumonia as are associated with an increased risk for lung cancer in Mexican Americans. Identifying risk factors such as *C. pneumonia* infections and COPD may induce targeted treatments to help attenuate progression to lung cancer. Furthermore, the observed increased risks in our study of Mexican Americans is similar to the increased risks to that of non-Hispanic whites which lends support to the hypothesis that once Mexican Americans are assimilated into US culture their rates of chronic diseases, including cancer, mimics rates observed among non-Hispanic whites [2, 5]. Our findings that Mexican Americans risk of lung cancer significantly differs from what we have previously published for African Americans [24] from the same multiracial/ethnic lung cancer case–control study may indicate an enhanced genetic predisposition for lung cancer in certain race/ethnicities. Therefore, the development of gene-environment interaction studies are needed to identify specific genes associated with this increased risk of lung cancer. Since this study is one of the largest case–control analyses assessing lung cancer risk among Mexican-Americans, we believe disseminating our findings is vital to address the gap that exists in Mexican American lung cancer research, while advancing our knowledge of the United States' fastest growing ethnic population.

Acknowledgments

This work was supported by the National Cancer Institute at the National Institutes of Health (K07CA093592 to C.J.E., CA55769 and CA127219 to M.R.S., and CA123208 and CA131327 to C.J.E.), and by a National Cancer Institute at the National Institutes of Health cancer prevention fellowship (R25T CA57730 to M.K.M.), Shine Chang, Ph.D., Principal Investigator.

References

1. American Cancer Society. Cancer facts and figures for Hispanics/Latinos 2009–2011. 2010. [cited 2010 January 19]; Available from: http://www.cancer.org/downloads/STT/F862309_final%20reduced.pdf
2. Eschbach K, Mahnken JD, Goodwin JS. Neighborhood composition and incidence of cancer among Hispanics in the United States. Cancer. 2005; 103:1036–44. [PubMed: 15672387]

3. Wiencke JK, et al. Lung cancer in Mexican-Americans and African-Americans is associated with the wild-type genotype of the NAD(P)H: quinone oxidoreductase polymorphism. Cancer Epidemiol Biomarkers Prev. 1997; 6(2):87–92. [PubMed: 9037558]
4. Smith BS, et al. Disparities in lung cancer stage, treatment and survival among American Indians and Alaskan Natives. Lung Cancer. 2011; 72:160–4. [PubMed: 20889227]
5. Cohen, B. Introduction, Chapter 1. In: Anderson, NB.; Bulatao, RB.; Cohen, B., editors. National Research Council (US) Panel on Race, Ethnicity, and Health Later in Life; Critical Perspectives on Racial and Ethnic Differences in Health in Late Life. Washington, D.C: National Academies Press; 2004.
6. American Cancer Society. Cancer facts and figures 2012. 2012. [cited 2012 March 2]. Available from: http://www.cancer.org/Research/CancerFactsFigures/CancerFactsFigures/cancer-factsfigures-2012
7. Alberg AJ, Samet JM. Epidemiology of lung cancer. Chest. 2003; 123(suppl):21S–49S. [PubMed: 12527563]
8. Mayne ST, Buenconsejo J, Janerich DT. Previous lung disease and risk of lung cancer among men and women nonsmokers. Am J Epidemiol. 1999; 149(1):13–20. [PubMed: 9883789]
9. Samet JM, Humble CG, Pathak DR. Personal and family history of respiratory disease and lung cancer risk. Am Rev Respir Dis. 1986; 134(3):466–70. [PubMed: 3752703]
10. Alavanja MC, et al. Preexisting lung disease and lung cancer among nonsmoking women. Am J Epidemiol. 1992; 136(6):623–32. [PubMed: 1442729]
11. Wu-Williams AH, et al. Lung cancer among women in north-east China. Br J Cancer. 1990; 62(6): 982–7. [PubMed: 2257230]
12. Brenner AV, et al. Previous pulmonary diseases and risk of lung cancer in Gansu Province, China. Int J Epidemiol. 2001; 30(1):118–24. [PubMed: 11171871]
13. Osann KE, Lowery JT, Schell MJ. Small cell lung cancer in women: risk associated with smoking, prior respiratory disease, and occupation. Lung Cancer. 2000; 28:1–10. [PubMed: 10704703]
14. McWhorter WP. Allergy and risk of cancer. A prospective study using NHANESI followup data. Cancer. 1988; 62(2):451–5. [PubMed: 3383143]
15. Skillrud DM, Offord KP, Miller RD. Higher risk of lung cancer in chronic obstructive pulmonary disease. A prospective, matched, controlled study. Ann Intern Med. 1986; 105(4):503–7. [PubMed: 3752756]
16. Santillan AA, Camargo CA Jr, Colditz GA. A meta-analysis of asthma and risk of lung cancer (United States). Cancer Causes Control. 2003; 14(4):327–34. [PubMed: 12846363]
17. Coussens LM, Werb Z. Inflammation and cancer. Nature. 2002; 420:860–7. [PubMed: 12490959]
18. Ballaz S, Mulshine JL. The potential contribution of chronic inflammation to lung carcinogenesis. Clin Lung Cancer. 2003; 5:46–62. [PubMed: 14596704]
19. Peek RMJ, Mohla S, DuBois R. Inflammation in the genesis and perpetuation of cancer: summary and recommendations from a National Cancer Institute—sponsored meeting. Cancer Res. 2005; 65(19):8583–6. [PubMed: 16204020]
20. Engels EA. Inflammation in the development of lung cancer: epidemiological evidence. Expert Rev Anticancer Ther. 2008; 8(4):605–15. [PubMed: 18402527]
21. O'Byrne KJ, Dalgleish AG. Chronic immune activation and inflammation as the cause of malignancy. Br J Cancer. 2000; 85(4):473–83. [PubMed: 11506482]
22. The Hispanic Population: 2010. 2011. [cited 2011 November 8, 2011]; Available from: http://www.census.gov/prod/cen2010/briefs/c2010br-04.pdf
23. Spitz MR, et al. A risk model for prediction of lung cancer. J Natl Cancer Inst. 2007; 99(9):715–26. [PubMed: 17470739]
24. Etzel CJ, et al. Development and validation of a lung cancer risk prediction model for African-Americans. Cancer Prev Res (Phila Pa). 2008; 1(4):255–65.
25. Tockman MS, et al. Airways obstruction and the risk for lung cancer. Ann Intern Med. 1987; 106(4):512–8. [PubMed: 3826952]
26. Schwartz AG, et al. Chronic obstructive lung diseases and risk of non-small cell lung cancer in women. J Thorac Oncol. 2009; 4(3):291–9. [PubMed: 19190518]

27. Littman AJ, et al. Prior lung disease and risk of lung cancer in a large prospective study. Cancer Causes Control. 2004; 15(8):819–27. [PubMed: 15456995]
28. Koshiol J, et al. Chronic obstructive pulmonary disease and altered risk of lung cancer in a population-based case-control study. PLoS One. 2009; 4(10):e7380. [PubMed: 19812684]
29. Raherison C, Girodet P. Epidemiology of COPD. Eur Respir J. 2009; 18(114):213–21.
30. Zhan P, et al. Chlamydia pneumonia infection and lung cancer risk: a meta-analysis. Eur J Cancer. 2011; 47(5):742–7. [PubMed: 21194924]
31. Koshiol J, et al. Lower risk of lung cancer after multiple pneumonia diagnoses. Cancer Epidemiol Biomarkers Prev. 2010; 19(3):716–21. [PubMed: 20200440]
32. Littman AJ, Jackson LA, Vaughan TL. *Chlamydia pneumoniae* and lung cancer: epidemiologic evidence. Cancer Epidemiol Biomarkers Prev. 2005; 14(4):773–8. [PubMed: 15824142]
33. Grayston JT, et al. A new respiratory tract pathogen: *Chlamydia pneumoniae* strain TWAR. J Infect Dis. 1990; 161(4):618–25. [PubMed: 2181028]
34. Hammerschlag MR. *Chlamydia pneumoniae* and the lung. Eur Respir J. 2000; 16(5):1001–7. [PubMed: 11153568]
35. Balkwill F, Mantovani A. Inflammation and cancer: back to Virchow? Lancet. 2001; 357:539–45. [PubMed: 11229684]
36. Preston-Martin S, et al. Increased cell division as a cause of human cancer. Cancer Res. 1990; 50:7514–21.
37. Hussain SP, Hofseth LJ, Harris CC. Radical causes of cancer. Nature. 2003; 3:276–85.
38. Talbot-Smith A, et al. Allergy, atopy, and cancer: a prospective study of the 1981 Busselton cohort. Am J Epidemiol. 2003; 157(7):606–12. [PubMed: 12672680]
39. Osann KE. Lung cancer in women: the importance of smoking, family history of cancer, and medical history of respiratory disease. Cancer Res. 1991; 51(18):4893–7. [PubMed: 1654203]
40. Cockcroft DW, et al. Is there a negative correlation between malignancy and respiratory atopy? Ann Allergy. 1979; 43(6):345–7. [PubMed: 517816]
41. Vena JE, et al. Allergy-related diseases and cancer: an inverse association. Am J Epidemiol. 1985; 122(1):66–74. [PubMed: 4014202]
42. Gabriel R, Dudley BM, Alexander WD. Lung cancer and allergy. Br J Clin Pract. 1972; 26:202–4. [PubMed: 4114639]
43. McDuffie HH. Atopy and primary lung cancer. Histology and sex distribution. Chest. 1991; 99(2): 404–7. [PubMed: 1989803]
44. Schabath MB, et al. Opposing effects of emphysema, hay fever, and select genetic variants on lung cancer risk. Am J Epidemiol. 2005; 161(5):412–22. [PubMed: 15718477]
45. Schabath MB, Gorlova OY, Spitz MR. Re: cancer mortality among US men and women with asthma and hay fever. Am J Epidemiol. 2006; 163(4):394–5. (author reply 395–6). [PubMed: 16371513]
46. Castaing M, et al. Is the risk of lung cancer reduced among eczema patients? Am J Epidemiol. 2005; 162(6):542–7. [PubMed: 16093291]
47. McCullough ML, Giovannucci EL. Diet and cancer prevention. Oncogene. 2004; 23(38):6349–64. [PubMed: 15322510]
48. Pope CA 3rd, et al. Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA. 2002; 287(9):1132–41. [PubMed: 11879110]
49. Siemiatycki J, et al. Controlling for potential confounding by occupational exposures. J Toxicol Environ Health A. 2003; 66(16–19):1591–603. [PubMed: 12959831]
50. Burnet M. Cancer; a biological approach. I. The processes of control. Br Med J. 1957; 1(5022): 779–86. [PubMed: 13404306]
51. Thun, MJ.; Henley, SJ. Tobacco. In: Schottenfeld, D.; Fraumeni, JF., Jr, editors. Cancer epidemiology and prevention. Oxford: Oxford University Press; 2006.
52. Haiman CA, et al. Ethnic and racial differences in the smoking-related risk of lung cancer. N Engl J Med. 2006; 354(4):333–42. [PubMed: 16436765]
53. Halpern MT, Gillespie BW, Warner KE. Patterns of absolute risk of lung cancer mortality in former smokers. J Natl Cancer Inst. 1993; 85(6):457–64. [PubMed: 8445673]

54. Tong L, et al. Lung carcinoma in former smokers. Cancer. 1996; 78(5):1004–10. [PubMed: 8780538]
55. Rothman, KJ.; Greenland, S. Modern epidemiology. Vol. 2. Philadelphia: Lippencott-Raven; 1998.
56. Hennekens, C.; Buring, JE. Epidemiology in medicine. Boston: Little, Brown & Company; 1987.
57. Koutros S, et al. Heterocyclic aromatic amine pesticide use and human cancer risk: results from the U.S. Agricultural Health Study. Int J Cancer. 2009; 124(5):1206–12. [PubMed: 19058219]
58. Blair A, et al. Reliability of reporting on life-style and agricultural factors by a sample of participants in the Agricultural Health Study from Iowa. Epidemiology. 2002; 13(1):94–9. [PubMed: 11805592]
59. Hoppin JA, et al. Accuracy of self-reported pesticide use duration information from licensed pesticide applicators in the Agricultural Health Study. J Expo Anal Environ Epidemiol. 2002; 12(5):313–8. [PubMed: 12198579]
60. Toren K, Brisman J, Jarvholm B. Asthma and asthma-like symptoms in adults assessed by questionnaires. A literature review. Chest. 1993; 104(2):600–8. [PubMed: 7802735]
61. Kehoe R, et al. Comparing self-reported and physician-reported medical history. Am J Epidemiol. 1994; 139:813–8. [PubMed: 8178794]
62. Giles WH, et al. The validity of self-reported hypertension and correlates of hypertension awareness among blacks and whites within the stroke belt. Am J Prev Med. 1995; 11:163–9. [PubMed: 7662395]
63. Vargas CM, et al. Validity of self-reported hypertension in the National Health and Nutrition Examination Survey III, 1988–1991. Prev Med. 1997; 26:678–85. [PubMed: 9327477]
64. Borrell LN, Crawford ND. Disparities in self-reported hypertension in Hispanic subgroups, non-Hispanic black and non-Hispanic White adults: the National Health Interview Survey. Ann Epidemiol. 2008; 18(10):803–12. [PubMed: 18922396]
65. Canales S, Ganz PA, Coscarelli CA. Translation and validation of life instrument for Hispanic American cancer patients: methodological considerations. Qual Life Res. 1995; 4:3–11. [PubMed: 7711688]
66. Checkoway, H.; Pearce, N.; Kriebel, D. Research methods in occupational epidemiology. 2. Oxford: Oxford University Press; 2004.

J Immigr Minor Health. Author manuscript; available in PMC 2014 October 01.

Table 1

Distribution of select characteristics of lung cancer cases and healthy controls in a Texas Case–Control Study of Mexican-Americans, 1991–2010

Characteristic	Cases (n = 204)		Controls (n = 325)		*P* value*
	No. or mean (SD)	%	No. or mean (SD)	%	
Gender					
Males	134	65.7	198	60.9	0.270
Females	70	34.3	127	39.1	
Mean age (years)					
Overall	63.5 (10.8)		60.9 (13.3)		0.013
Males	63.8 (10.4)		61.8 (12.9)		0.127
Females	63.0 (11.7)		59.4 (13.8)		0.065
Smoking status					
Never	43	21.1	110	33.9	
Former	72	35.3	118	36.3	0.001
Current	89	43.6	97	29.9	
Current smokers					
Smoking duration	43.4 (12.1)		34.5 (16.2)		<0.001
No. of cigarettes/day	23.3 (13.8)		13.5 (11.1)		<0.001
Pack-years: mean(SD)	50.7 (34.3)		25.1 (22.9)		<0.001
Smoking Intensity					
Light smoker: 0<pack-years ≤ 20	13	14.6	47	51.1	
Moderate smoker: 20<pack-years ≤ 40	24	27.0	23	25.0	<0.001
Heavy smoker: 40<pack-years	52	58.4	22	23.9	
Former smokers					
Smoking duration	37.9 (15.5)		24.6 (14.7)		<0.001
No. of cigarettes/day	20.8 (13.0)		16.6 (14.6)		0.045
Pack-years: mean	41.2 (32.0)		22.4 (31.6)		<0.001
Years since smoking cessation	10.6 (9.19)		16.2 (12.3)		0.001
Age at smoking cessation	57.5 (12.2)		44.7 (14.3)		<0.001
Smoking intensity					
Light smoker: 0<Pack-years ≤ 20	21	29.2	76	67.3	
Moderate smoker: 20<Pack-years ≤ 40	21	41.7	21	18.6	<0.001

Characteristic	Cases (n = 204)		Controls (n = 325)		*P* value*
	No. or mean (SD)	%	No. or mean (SD)	%	
Heavy smoker: 40<pack-years	30	29.2	16	14.2	
Co-morbidities					
Asthma	14	7.6	19	6.5	0.631
Chronic obstructive pulmonary disease[a]	42	22.8	34	11.6	0.001
Hay Fever	10	5.6	39	13.3	0.007
Pneumonia	46	25.3	35	11.9	<0.001
Histology					
Adenocarcinoma	80	42.3			
Squamous cell	50	26.5			
Other NSCLC	25	13.2			
Small cell	16	8.5			
Other LC	18	9.5			
Stage					
I	14	15.7			
II	5	5.6			
III	25	28.1			
IV	45	50.6			
Family history among first degree relatives					
Lung cancer	15	7.4	25	7.7	0.886
Smoking-related cancers[b]	53	26.0	81	24.9	0.785

SD standard deviation

*P values were derived from Pearson's χ^2 test for categorical variables and Student's t test for continuous variables

[a]COPD included emphysema and chronic bronchitis

[b]Smoking related cancers (e.g., head and neck; urinary bladder and kidney; esophageal; pancreatic; cervical; and esophageal)

Table 2

Relationship of smoking-related covariates and prior respiratory disease with lung cancer risk in a Texas Case–Control Study of Mexican Americans, 1991–2010

Variable	No. of cases	(%)	No. of controls	(%)	Multivariate odds ratio[a]	95 % confidence interval
Smoking status						
Never	43	21.1	110	33.9		
Former	72	35.3	118	36.3	1.6	1.0, 2.6
Current	89	43.6	97	29.9	2.4	1.5, 4.0
Current smokers						
Smoking duration						
≤30 years	15	16.9	30	32.6		
>30 years	74	83.1	62	67.4	1.9	0.8, 4.5
No. of cigarettes/day						
≤20 per day	58	65.2	81	87.1		
>20 per day	31	34.8	12	12.9	3.7	1.7, 8.1
Smoking intensity						
Light smoker: 0<pack-years ≤ 20	13	14.6	47	51.1		
Moderate smoker: 20<pack-years ≤ 40	24	27.0	23	25.0	4.2	1.7, 10.3
Heavy smoker: 40<pack-years	52	58.4	22	23.9	10.0	4.0, 24.7
Former smokers						
Smoking duration						
≤30 years	25	34.7	70	62.0		
>30 years	47	65.3	43	38.0	1.8	0.9, 3.7
No. of cigarettes/day						
≤20 per day	43	59.7	93	82.3		
>20 per day	29	40.3	20	17.7	3.3	1.5, 7.1
Smoking intensity						
Light smoker: 0<pack-years ≤ 20	21	29.2	76	67.3		
Moderate smoker: 20<pack-years ≤ 40	21	41.7	21	18.6	3.6	1.6, 8.33
Heavy smoker: 40<pack-years	30	29.2	16	14.2	5.8	2.4, 13.9
Years since smoking cessation						
≤10 years	42	58.3	49	41.5		

Variable	No. of cases	(%)	No. of controls	(%)	Multivariate odds ratio[a]	95 % confidence interval
>10 years	30	41.7	69	58.5	0.4	0.2, 0.7
Age at smoking cessation						
≤30 years	2	2.8	25	21.2		
>30 years	70	97.2	93	78.8	5.8	1.2, 27.4
Smoking cessation						
Years>10, Age ≤30	2	2.8	22	18.6		
Years<10, Age ≤30	0	0.0	3	2.5	–	–
Years>10, Age>30	28	38.9	47	39.8	3.7	0.7, 18.8
Years<10, Age>30	42	58.3	46	39.0	8.2	1.6, 40.6
Respiratory co-morbidities						
No respiratory disease	115	57.2	224	68.9		
Any respiratory disease	86	42.8	101	31.1	1.5	1.0, 2.3
No Asthma	184	92.5	305	93.8		
Asthma	15	7.5	20	6.2	1.3	0.6, 2.8
No COPD	154	77.0	285	88.0		
COPD	46	23.0	39	12.0	2.0	1.2, 3.3
No hay fever	184	94.4	284	87.7		
Hay fever	11	5.6	40	12.3	0.4	0.2 0.9
No pneumonia	148	75.1	286	88.0		
Pneumonia	49	24.9	39	12.0	2.2	1.3, 3.6

[a] All variables are adjusted for age, sex, and self-reported pesticide exposure. Respiratory co-morbidities are also adjusted for smoking intensity (number of smoking years/number of cigarettes per day)